Dead space and its components

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This chapter is most relevant to Section F6(iv) from the 2023 CICM Primary Syllabus, which expects the exam candidates to be able to *"define dead space and its components"*. This has come up twice in the past papers:

- Question 3 from the first paper of 2018
- Question 7(p.2) from the first paper of 2010

Each time the components of dead space were asked about. Occasionally the candidates are asked about the different ways of measuring dead space. Occasionally, about how increasing the dead space volume affects respiratory function. So far, nobody has been asked about the physiological factors which affect dead space, but surely that time will one day come.

In summary:

- Dead space is the fraction of tidal volume which does not participate in gas exchange.
- It is composed of apparatus dead space and physiological dead space:
- **Apparatus dead space** is the dead space in an artifical breathing circuit
  - It can increase the total dead space:
    - Mechanical ventilation using a large NIV mask
    - Large circuit components, eg. a big HME
  - It can reduce the total dead space
    - Use of ETT (smaller volume than the upper airway)
    - Tracheostomy (bypass the upper airway altogether)
- **Physiological dead space** is measured using the Enghoff modification of Bohr's equation, using arterial CO_2 instead of exhaled CO_2, and it is composed of:
  - **Alveolar dead space**
    - The difference between the physiological dead space and the anatomical dead space
    - This is the volume of gas which fills lung units which are unperfused or poorly perfused
    - Wests' Zone 1 contain alveolar dead space.
    - Under normal circumstances, this volume is minimal.
  - **Anatomical dead space**

        - Measured by Fowler's method
        - Represented by Phase I and half of Phase II in the single-breath nitrogen washout test.
        - Represents the volume of gas in the conducting airways
    - **Shunt,** when massive, can produce the illusion of increased Enghoff dead space because it increases arterial CO_2

The single best peer-reviewed resource for this topic is probably Roberts (2015). For a nice historical perspective, one may lose oneself in Robert Klocke's essay "Dead space: Simplicity to complexity" (2006). If one can get a hold of it, the article by Hedenstierna & Sandhagen (2006) is also valuable because it reaches beyond the normal anatomical-alveolar-apparatus trichotomy to find weird new dead spaces all over the place. In order to access it (as it will not show up in normal search results) one needs to dig it out of the *Minerva Anaesthesiologica* archive, where it is freely available to anybody who can answer a CAPTCHA.

Definition of dead space

Ward S. Fowler (1948), as a part of an introduction to his article, wisely counsels that *"since the terminology used by various writers is not uniform, it seems advisable to clarify the meaning of dead space."* This advice remains current. Though, like obscenity, everybody knows it when they see it, when asked to formally define dead space many people will actually give a definition for one of its components, usually anatomical dead space (this is true even of published authors).

The author of term is generally believed to be Christian Bohr (1891), who referred to it as *"schädlichen Raumes"* (literally "the harmful space"). The first time it referred to as "dead" was apparently by Haldane in 1895. Neither of these early pioneers had given much thought to the fact that people would have to remember this thing for their exams, and so nobody really focused on making a short pithy definition. In fact nobody had even settled on an official name to call it, as sixty years later Rossier & Bühlmann (1955) were still complaining that

> *"despite the acceptance of the term 'dead space' by most physiologists, there are nevertheless some who today still call it 'noxious space' ('espace nuisible,'*

> *'schadlicher Raurn'), 'volumen inefficax' etc."*

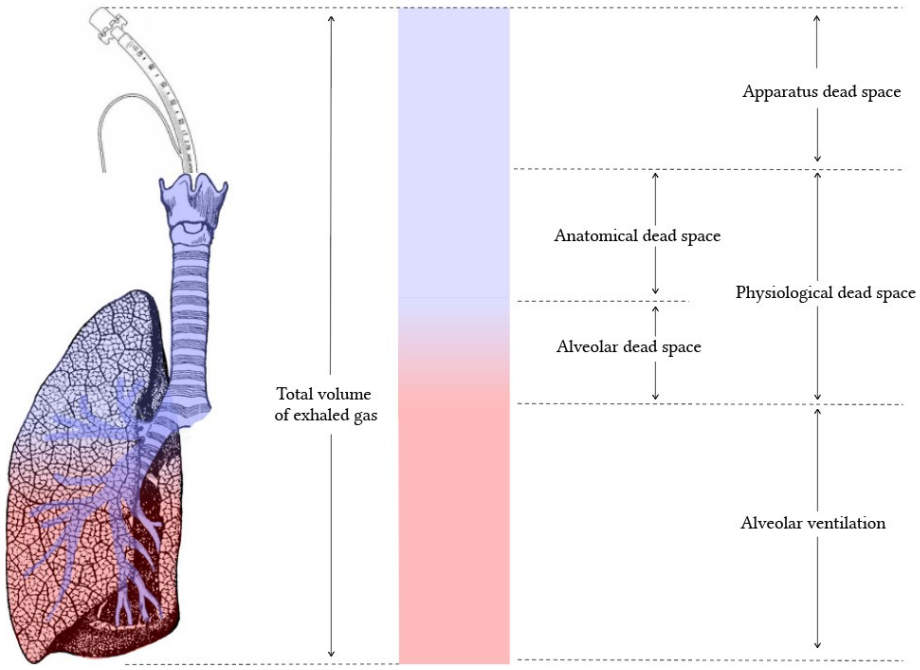
Fortunately, the summary paragraphs from *Nunn's* (8th ed) give an excellent one-liner which is definitive as far as CICM First part preparation is concerned:

> *Dead space is the fraction of tidal volume which does not participate in gas exchange.*

This is a perfect way of wording it, because takes into account the usual proportional relationship of dead space and minute ventilation, where:

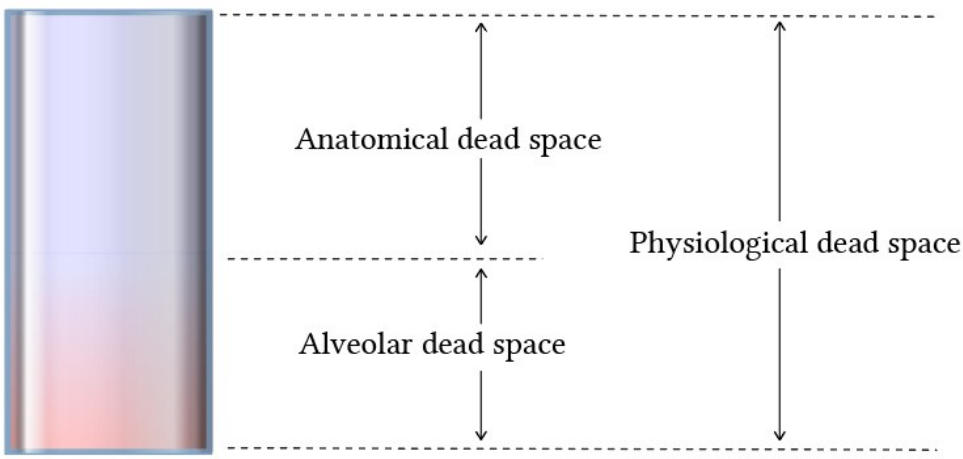
> Alveolar ventilation = Resp rate × (tidal volume - dead space)

In short, "alveolar ventilation" is the volume of gas which did something useful on its way through the lungs, and dead space ventilation is the volume which *did not.* This volume of useless gas has three main components: apparatus dead space, anatomical dead space and alveolar dead space. There are a few others, but they occupy a weird fringe at the borders of the passing mark, and unless one really wants to wreck the CICM grading bell-curve one really does not need to think about them. For visual learners, one could represent these dead spaces in the form of an illustration:

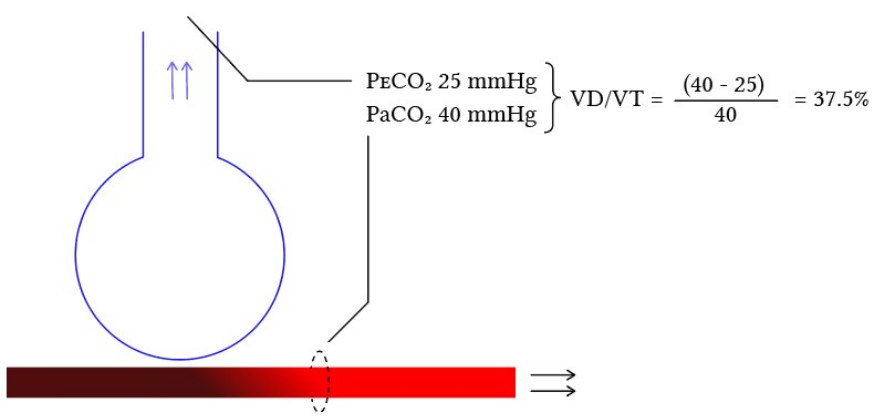


Physiological dead space

Physiological dead space is the combination of the anatomical and alveolar dead space components. Rather than representing it as a volume in ml/kg, it is better to represent it as a fraction of the tidal volume (or minute volume), largely because it tends to change together with the tidal volume. The normal values for this tend to differ across textbooks, but they all seem to fall into the ballpark of 20-30%. Henrik Enghoff's original 1938 paper gave 34% as the average value.



From a functional standpoint, physiological dead space is the thing you're measuring using the Enghoff modification of the Bohr equation, which is essentially just the difference between arterial and mixed expired pCO_2 divided by the arterial pCO_2. Here is a visual representation of this, for an example which will be used later:



Note that the $PECO_2$ is not the *end tidal* pCO_2, but the *mixed expired* pCO_2. The difference is quite substantial. The mixed expired pCO_2 is the value you'd get if you collected all the expired gas into a bag, where it could mix vigorously and become homogeneous. For a normal person, the mixed expird pCO2 is usually something like 25 mmHg (Hansen et al, 2007)

So anyway: the physiological is a virtual space, produced by the need to simplify the discussion of the lung as being composed of two compartments, one of which is completely unperfused. This oversimplification leads down weird avenues, for example where the presence of shunt can produce the appearance of dead space. However, it is generally accepted as a solid way to approximate the dead space fraction because it is so convenient (arterial blood gases being widely available), in contrast to other methods.

Anatomical dead space

Of the dead space compartments, this one was discovered first, probably because the concept of alveolar dead space is more difficult to intuit than the idea that the trachea exchanges no gas. Thus, by 1882, Nathan Zuntz was able to measure the conducting airway volume in cadavers, and propose that this fraction of the tidal volume probably plays no role in ventilation.

Nunn's (8th ed), in a quick-revision summary section, defines anatomical dead space as:

> *"the volume of the air passages through which the gas is conducted to the alveoli"*

In short, this volume encompasses from the lips and *vibrissae* to the innermost terminal bronchioles. This definition, though anatomically descriptive, fails to describe the relationship between the two components of physiological dead space. It would probably be better to define it as

> *"the volume of gas exhaled before the CO_2 concentration rises to its alveolar plateau"*

This is also from Nunn's. It is a good functional definition, but it somehow fails to capture the way the conducting airways exchange no gas, which is a pretty

central element. Also, by the time the *CO_2 concentration rises to its alveolar plateau,* one is no longer talking about dead space, i.e. the definition would encompass both physiological and anatomical dead space. At risk of adding another pointless definition, one possible way to reword this would be:

> *"Anatomical dead space is the portion of dead space which is external to the alveoli, consisting of mainly conducting airways, and represented by Phase I and half of Phase II in the single-breath nitrogen washout test"*

Anyway. Given the name, one might surmise that the volume of anatomical dead space is probably influenced by some anatomical factors. These are:

- **Body size and age:** The anatomical dead space is said to stay about 2ml/kg of ideal body weight for your entire adult life, but in the infant it is larger proportionally to body weight, around 3ml/kg in early infancy (Numa & Newth, 1996). Most of the age-related change in anatomical dead space is due to changes in the extrathoracic airways: the conducting airways of the lungs themselves grow together with the body at a pretty linear rate (1ml/kg). Having said all this, the actual relationship between body size and anatomical dead space appears to be fairly erratic, and when applying these ml/kg values in real life one is usually wrong by a margin greater than 25% (Brewer et al, 2008).
- **Posture:** Intrathoracic anatomical dead space decreases as lung volume decreases, and so can be expected to be slightly smaller with the subject in a supine position because all the volumes are decreased when you are supine. *Nunn's* gives *"a third less than when sitting"* but no reference. This may be due to some sort of confusion; certainly *physiological* dead space (anatomical plus alveolar) decreases by a third when a seated subject is made supine (Fowler, 1950). The change in *anatomical* dead space is probably much less than that; Riley et al (1959) estimated that it was about 8ml for one of their subjects.
- **Airway manoeuvres:** a jaw thrust and chin lift adds an extra 40ml or so (Nunn et al, 1959), and conversely letting the patient's unconscious head flop uselessly with their chin to their chest will rob you of about 30ml of dead space. From a practical perspective, it is probably pointless to know this, because of how meaningless this decrease in dead space would be in an apnoeic patient with an obstructed airway.

- **Lung volume:** because as the lung inflates, neighbouring alveoli will pull on the smaller airways by traction, and increase their diameter. This might be some trivial change for any single given bronchiole, but because there are thousands of them, it all builds up. Therefore, there is some variability of the anatomical dead space over one's vital capacity, though it is not as great as the variability of the alveolar dead space.
- **Bronchospasm:** One would expect for something like asthma to decrease the anatomical dead space volume, purely by decreasing the internal diameter of the airways. However, at least when measured by conventional methods, it does not appear to do so (at least not in asthmatic children, according to Kerr et al, 1976). However, bronchoconstriction is mentioned in most textbooks as one of the factors which affect anatomical dead space, usually with no direct reference.
- **Bronchiectasis:** Logically, if bronchial constriction should decrease the anatomical dead space, then surely bronchial *dilatation* should increase it. However, among resting patients with cystic fibrosis, Thin et al (2004) were not able to demonstrate any appreciable increase in dead space volume when they were compared to healthy controls.

Anatomical dead space is also affected by flow characteristics during respiration, which is cheating in a way. Though under these circumstances the actual size and shape of the conducting airways remains largely the same, the changes in gas flow can produce situations where the apparent dead space volume has changed. This can happen during ventilation with low tidal volumes, or where the subject is performing a breath hold, or where high flow nasal prongs are involved.

The effect of a low tidal volume on anatomical dead space

When one takes tidal volumes below the expected volume of anatomical dead space (i.e. a V_T less than 150ml in an adult), one might expect that all of the tidal volume is just dead space, and that no alveolar ventilation can possibly occur. That, in fact, is not the case. When measured by Bohr's method, the anatomical dead space appears to decrease in proportion to the tidal

volume. Nunn & Hill (1959) found that the threshold for this is around 350ml. In fact with small enough tidal volumes, they were able to sample alveolar-looking expiratory gas from the carina of one of their subjects, suggesting an intrathoracic anatomical dead space volume of *0ml*.
This is because of two main phenomena:

- **Laminar flow:** with low flow rates, there is less turbulent flow in the airways, and a relatively small central column of gas can move in and out of the alveoli while the peripheral gas in the conducting airways remains undisturbed, clinging to their walls. In normal breathing this already occurs in the smaller airways (distal to Generation 10) because the flow slows to a crawl down there, but with low tidal volumes this probably includes large airways as well.
- **Expiratory gas mixing:** because of the relatively slow movement of gas out of the alveoli, there is more chance for the alveolar gas to mix (by diffusion) with the gas of the conducting airways, making the exhaled gas concentration more homogeneous and alveolar-like, which has the effect of making the anatomical dead space look smaller.

By extension of the same thought as above, if one holds one's expiratory gases in one's conducting airways, given some time they will all mix by various means (cardiac pulsation agitating them, diffusion, etc etc). The outcome will be that, upon exhaling, one's larynx will contain a gas mixture which will resemble one's alveolar gas. The measurement of dead space by analysis of expiratory gases will therefore yield bizarre results, with anatomical dead space appearing confined to the volume of the supraglottic structures.

Alveolar dead space

In the briefest way possible, alveolar dead space is all the dead space which is not anatomical. It is remarkable that such a simple concept should produce so much confusion, and the level of confusion seems to escalate the deeper one digs into the literature. The worst thing one could do is try to compare different authors' interpretations to establish one which is superior. For example, to borrow from *Nunn's,* alveolar dead space volume is:

> *"the part of the inspired gas that passes through the anatomical dead space to mix with gas at the alveolar level, but which does not take part in gas exchange."*

To word it slightly differently (*part of the inspired gas* might not sound scientific enough for some readers) the alveolar dead space volume can be defined as the *fraction of the tidal volume* which interacts with alveoli without any gas exchange taking place. Realistically of course this is a composite of gas coming back from truly unperfused alveoli together with gas coming back from alveoli which were merely *under*perfused, but it is convenient to pretend that this dead space volume is a sharply demarcated block of gas. If one is seduced by simplicity, one would hold to the definition that:

> Alveolar dead space is the volume of gas which ventilates lung units with V/Q = ∞

This is good enough for people who teach other people about lung physiology for a living, and it is also convenient because the Bohr and Enghoff methods of measuring dead space end up reporting a single volume which indeed has a V/Q = ∞. However, most reasonable people would probably want their definition to recognise that there are these shadowy regions of V/Q maldistribution, in which case:

> Alveolar dead space is the volume of gas which ventilates lung units with V/Q > 1.0

Weirdly, other authoritative sources (eg. this article on PE by Goldhaber & Eliott, 2003) describe lung units with V/Q = ∞ as "anatomical dead space", and claim that anatomical dead space increases with PE, whereas "alveolar dead space" is presumably all those other lung units which have a V/Q ratio between 1.0 and infinity, including those whose V/Q ratio is immeasurably high but *not* infinite. Presumably, because these people got published in *Circulation*, some senior peer reviewer somewhere agreed that this is a well-established definition for these terms.

In summary, there does not appear to be consensus on what alveolar dead space is, or how to represent it in a human-readable sentence. From the abovestated gibberish, fragments of reason can be extracted to make memorable soundbites for the vivas:

- Alveolar dead space is the volume of gas which fills lung units which are underperfused / not perfused / not participating in gas exchange (pick the description which produces the fewest furrows in the examiner's brow)
- It is the difference between physiological dead space and anatomical dead space.
- Wests' Zone 1 contain alveolar dead space.
- Under normal circumstances, this volume is minimal.

There are several factors which affect the size of this volume:

- **Poor cardiac output:** in a normal healthy person the size of the alveolar dead space is minimal, because the cardiac output pumps enough to perfuse even the most forgotten apical alveoli. However, if one's cardiac output is poor (i.e if the right ventricle is unable to generate much systolic pressure), the perfusion of these iffy alveoli will decline and dead space will increase. Probably the most relevant example of this scenario is haemorrhagic shock. Steenblock et al (1976) exsanguinated a bunch of dogs to the point of near death (cardiac output down to 20-30%) and demonstrated that their dead space ventilation increased significantly, from 98.2 to 240 ml/min/kg.
- **Parenchymal lung disease:** destructive processes such as emphysema increase dead space by eating up the well-perfused alveoli and leaving behind empty spaces bounded by unperfused scar tissue Helmy et al (2015) found that the mean alveolar dead space in their cohort of unweanable COPD patients was about 52%.
- **High positive pressure ventilation:** increasing the positive pressure pushes blood out of the well-ventilated regions and therefore increases the alveolar dead space. Gogniat et al (2018) determined that increasing the PEEP from 0 to 16 in ARDS patients can increase their dead space from around 44% to around 51%.
- **Pulmonary vascular occlusion:** obviously, if you block the pulmonary arteries with something, you will reduce the perfusion of the downstream lung units, and turn those lung units into alveolar dead space. One of the conventional ways of doing this is with a venous thromboembolism. Burki (1986) found the total dead space of patients with a radiologically detectable PE was almost always over

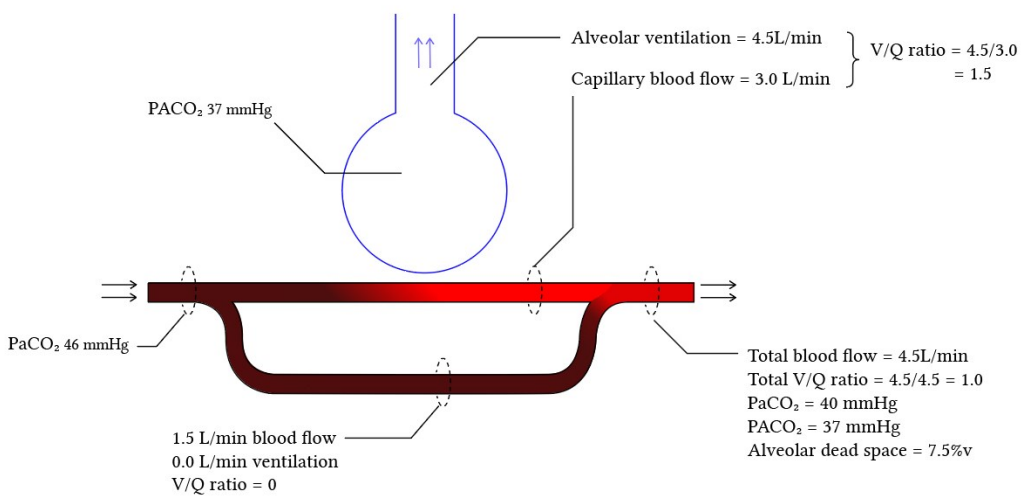
40%, making it relatively sensitive as a diagnostic test.

- **Posture:** given that alveolar dead space is generally attributed to poor perfusion, and given the known tendency of gravity to affect pulmonary blood flow, it would be logical to expect changes of posture to have an effect on dead space volume. One would expect it to decrease with the subject supine, for example. This expectation was confirmed by Riley et al (1959), who determined that the majority of this change (the magnitude of which was about 83ml on average) was due to changes in the alveolar dead space. Bjurstedt et al (1962) got 28ml in a slightly different experimental setup (they used healthy young males, whereas Riley et al experimented on each other).
- **Extremes of gravity or acceleration:** Following a logical extension of the above, if gravity plays such a major role in determining the alveolar dead space fraction, then alveolar dead space should be minimal under conditions of weightlessness. That is indeed what Prisk et al (1995) found: astronauts from Spacelab 1 and 2 had a measurable decrease in their alveolar dead space, down by 30% from standing values and down by 15% from supine. Conversely, high gravity and acceleration should suck blood out the fore lung regions and push it aft. To demonstrate this, Von Nieding et al (1973) exposed a group of unfortunates to "hypergravitational stress" by shoving them into a centrifuge spinning up to 4G. As expected, the subjects became distressed and their gas exchange failed in a manner consistent with increased dead space ventilation (lower oxygen and rising CO_2 in spite of increasing respiratory rate), until measurements could no longer be performed because *"at this time it was no longer possible to get blood from the hyperemized ear lobe, indicating a failure of blood supply to the peripheral tissues"*
- **Open chest and one lung ventilation:** If the surgeon opens the hemithorax and you have failed to isolate and deflate the ipsilateral lung, it will inflate with positive pressure breaths and herniate comically out of the wound, to your considerable embarrassment. This lung will also be made up of predominantly Zone 1 units, and will therefore meet at least one of the definitions of alveolar dead space. It is relevant to know this random fact because the ANZCA exams are often a source of inspiration for the CICM examiners, who are themselves anaesthetists, and so this anaesthesia-specific element

might one day sneak into the CICM First Part exam. It is also worth knowing that, insofar as 15 minutes of Googling can determine, there is no experimental reference for this assertion, and the only source which constantly comes up is a review of one-lung ventilation by Lohser & Ishikawa (2011). The *contra*lateral lung can definitely develop dead space in spite of the fact that it is dependent and should theoretically receive better perfusion; however, as Tusman et al (2015) point out, it is still possible to dead-space that good lung by ventilating it with punishingly large tidal volumes.

Dead space due to right-to-left shunt

The presence of a large enough shunt can give rise to the appearance of increased physiological dead space, provided one uses the Enghoff definition of dead space. Observe, a clever example from Hedenstierna & Sandhagen (2006), rendered into puerile diagrams:



In this thought experiment, the dead space fraction of the tidal volume is 7.5%. However, one should note that in this one-alveolus model, there are no unperfused lung units. Thus, where shunt is substantial, it can create the illusion of alveolar dead space because it increases the difference between the alveolar and arterial CO_2. The reader is warned: this is only an illusion, resulting from a limitation of the Bohr-Enghoff equation. There's no unperfused alveoli in there. Authors such as Hedenstierna

& Sandhagen (2006) even warn against giving this thing a name like "shunt dead space" because it *"may give the reader the wrong impression that we do deal with a real dead space"*. It does, however, behave very much like dead space, in the sense that increasing the ventilation will have little additional effect on decreasing the $PaCO_2$.

For this scenario to occur, the shunt has got to be pretty big (in the scenario above, one third of all cardiac output is going through the shunt). In real life, shunts like this are occasionally seen in the setting of ARDS. Nicklason et al (2008) modelled some shunt fraction scenarios and determined that with a normal cardiac output, a shunt fraction of 60% would be expected to produce an alveolar dead space fraction of around 21%. Moreover, even at a more modest shunt fraction of 40%, decreasing the cardiac output from 5L/min to 3 L/min (totally plausible in the ICU) increased the alveolar dead space from 11% to 16%.

Apparatus dead space

The effect of airway equipment on changing the dead space is discussed in greater detail in the chapter on the effects of positive pressure ventilation. In summary, the main reason for the change is that under some circumstances the aforementioned equipment will either decrease the anatomical dead space by bypassing the upper airway structures, or add to it by adding extra volume in the form of circuit components.

changes in anatomical dead space with mechnical ventilation

In this fashion, intubation or tracheostomy decrease anatomical dead space by up to 50%, whereas NIV increases anatomical dead space by the volume of the mask, or about 50ml (Saatci et al, 2004).

Eponymous dead spaces

Just as a reader rightly wonder how many more dead spaces they could possibly tolerate, this chapter confronts them with several more.

References to these are seen throughout the literature, and there is probably some merit in discussing them. In short, they are named after researchers who first demonstrated some novel way of measuring dead space, and the volumes they describe are dead space as measured by the eponymous method. As such, each dead space has some boundaries and inaccuracies which mirror the limitations of their specific measurement technique, discussed in greater detail in the chapter on dead space measurement. Here, a brief summary will suffice:

- **Bohr dead space:** The dead space measured by Bohr's method, using the alveolar CO_2 concentration, which generally corresponds to the physiological dead space in normal healthy people. Difficult to measure, as the mean alveolar CO_2 is difficult to precisely determine, and as individual alveoli will have very different CO_2 values. Unaffected by the presence of a shunt.
- **Enghoff dead space,** which is the dead space measured by Enghoff's modification of the Bohr equation, using arterial CO_2 and therefore inviting error in the shape of shunt. In most textbooks, the Enghoff space is synonymous with physiological dead space.
- **Fowler dead space** usually refers to anatomical dead space, because the Fowler method is the classical means of measuring this space (although it can also be used to determine the closing capacity). As it does not have anything to do with CO_2, this method does not get confused when a huge shunt is present.

Previous chapter: Effects of ventilation-perfusion mismatch on gas exchange

Next chapter: Measurement of dead space

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